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ORIGINAL ARTICLES.

ON THE RETROGRESSION OF RETINAL GLIOMA.*

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In the work of Leber on the diseases of the retina, recently published, there is an illustration of a case of retinitis exudativa (p. 1276) in the shape of a so-called pseudoglioma in a 5 years old boy with the remark, "glioma with a benign course can probably be excluded." No clinician is likely to contradict this remark. For even, if in exceptional cases the first stage of the disease may last five years,[1] still during this period the course of these cases cannot be called a benign one, since the tumor from the smallest beginnings in slow but continuous progress had grown so far that the affection finally entered the second stage, that of increased intraocular tension, and then quickly became fatal. Graefe, in his publication on glioma,[2] said, "I feel like declaring myself emphatically against the view that there are in the course of these diseases longer periods, even of years, in which it is said to remain quiet. Moreover, I do not think that glioma is a tumor which is capable of retrogression."

If to-day I report a case of undoubted glioma, in which I have seen in five years not only no progress in the tumor formation, but on the contrary, lately an undoubted retrogression, this communication must prove of decided interest.

On July 20th, 1910, the 4 years old boy, A. W., was brought by his parents to my consulting rooms with the statement that the

*Centrabl. f. prakt. Augenhlk., May, 15.

1. Wintersteiner. Neuroepithelioma, 1897.
2. Arch., XIV, 2, p. 155.

child's right eye had shown a divergence ever since birth. The eye had always looked different from its fellow, although they could not say wherein lay the difference. They had, perhaps, sometimes seen something gray in the eye, and according to their opinion the eye had always been blind. During the last few months more and more a yellow shining appearance had been observed. In the left eye the parents had noticed nothing abnormal.

The well developed, lively child keeps the right eye with normal motility always in a strongly divergent position. The anterior part of the globe is normal. With oblique illumination an intensely yellow reflex is seen to come from the interior of the eye, which comes from the detached retina which lies close to the posterior surface of the lens. It appears thickened and in the yellow mass of degenerated tissue numerous bloodvessels are seen.

The diagnosis of glioma could not be doubted. The frequency of its occurrence in both eyes prompted me to make a thorough examination of the other eye with which the child saw apparently well. As soon as it was illuminated by the ophthalmoscope a change from the normal red fundus reflex into a yellow one made certain the presence of circumscribed nodules of tumor tissue. The largest one of these was situated in and down from the papilla. It began with a sharply defined convex margin at a distance of about 6 P.D. from the edge of the papilla and reached a width of several P.D. toward the periphery; upwards it was about 3 P.D. large. The tumor protruded far into the vitreous body and contained large tortuous bloodvessels. A second much smaller, yellowish white nodule reaching far forward into the vitreous body, lay below the first one. A third very small nodule of the same appearance was situated in the lower external half of the fundus.

The papilla, moreover, presented a noticeable appearance since the bloodvessels going downward, arteries as well as veins, were very thick and tortuous.

Although the examination was made very difficult by the continued movements of the child and therefore no more accurate details could be described, the diagnosis of glioma seemed absolutely certain from what was found. I had to explain the seriousness of the case to the parents. As is easily understood, they refused the enucleation of both eyes. They would rather let the child die than to have the seeing eye of the lively child, which

played happily around them, removed. In order to do something therapeutical we used Roentgen rays on the eyes on July 26th and 27th. Aside from this, potassium iodide was given internally and a bathing cure pursued at Hall. On September 5th, 1910, no change was visible in either eye. On September 11th the patient was brought back, because there had for several days been severe pains in the right eye, which was inflamed. This eye now showed all the signs of a high increase in intraocular pressure and the parents themselves demanded now its removal. This was done on the next day by Dr. Denk at Linz. The eyeball was kindly given to me for examination. When I saw the child again on June 28th, 1911, the condition of the left eye was unchanged. Half a year later—December, 1911—I examined the child again and I had the impression that the tumors had increased somewhat in size. However, during 1912 I could find no change in the nodules. The child then disappeared from my observation and I thought it had long ago died from its disease when some time ago, at the end of March of this year, he appeared with his parents in my office, grown to a strong boy. The child is bodily excellently developed, goes with success to the public school and does not complain of any difficulty in his left eye. In the right orbit, which had remained free from relapses, was an excellent artificial eye, so that the lack of the real one could hardly be detected. The eye, which now can be tested, has normal visual acuity and is externally normal. The papilla shows nothing peculiar; the blood vessels are no longer broad. The focuses in the retina persist in the same form and size as formerly, but they are flattened, almost like atrophic choroiditic foci, from which they are, however, distinguishable by their consisting of crumby, chalk-like white masses. Especially the small nodule consists of these white crumbs, which lie in a dark gray pigmented area. The numerous, thick and tortuous bloodvessels previously seen in the nodules have almost disappeared. The histological findings in the enucleated right eye show a typical glioma exophytum. The retina, in toto detached reaches from the papilla forward to the posterior lens surface, which by the pressure of the protrusion in the retina is deformed in the well-known manner. From the retina the tumor has grown into the subretinal space and filled this partially. The retina itself is almost altogether changed into tumor tissue, only here and there the nuclear layers can be made out as the last remnants of its original structure. From the subretinal

tumor numerous particles have fallen on the choroid and there begun to grow (dissemination). The cells of the tumor are the usual small round cells which are well stained only around the bloodvessels, while somewhat farther from these the necrosis of the cells is apparent. No rosettes were found. Otherwise the eye shows the usual signs of secondary glaucoma in a tumor eye; pressing forward of the iris and lens, extensive peripheral anterior synechia, high-grade atrophy of the whole uveal tract, atrophy and excavation of the optic nerve. The external bulbus capsule has as yet nowhere been attacked by the tumor.

First, a few general remarks about this case, especially about the length of time the disease existed. At birth already the parents noticed the divergence of the right eye, an almost certain sign that even at that time the retina was attacked by the tumor formation. In spite of this the histological examination of the eye enucleated during the fourth year of life of the child did not show very far progressed neoplastic changes. The retina is taken up by it and the tumor reaches somewhat into the subretinal space. The other tissues of the eye are still intact and the secondary nodules on the choroid due to dissemination are still in the first beginning. Such a slow growth has repeatedly been observed.[3] How long the left eye contained nodules, we have no data to judge. From the slow growth they may have been present for years, and perhaps were, also, congenital. Wintersteiner's[4] opinion that glioma is always a congenital affection, even in the cases in which it becomes clinically manifest in the second decennium only, is very probably correct. The neoplastic process in our case showed not only a noticeable standstill, but definite signs of retrogression. The latter is shown especially by the bloodvessels of the papilla, which ran in the direction of the nodules, becoming again normal, while previously they were very much enlarged and tortuous. It is probably a sign of retrogression that the nodules assumed rather the appearance of choroiditic depigmented foci, partly with pigment proliferation; but this must not be judged to be a proof of the disappearance of all tumor tissue, since similar pictures have been seen in growing glioma which was confirmed by histological examination.[5] However, another point merits attention—the uniform and simultaneous retrogression of the tumor formation in all three nodules. It would be easily understood that in one nodule, for

3. Wintersteiner. A. A. O., p. 158.
4. Page 115.
5. Hosch. A. f. A., XVIII.

instance, by circulatory disturbances a more considerable necrosis would result than usual in such small nodules, which accidentally might lead to the destruction of all tumor cells; but this can hardly be the explanation of the simultaneous changes of the same degree in all three nodules. Only a simultaneously acting agent, perhaps present in the blood, could be sufficient to produce such alterations.

When one speaks of retrogression or even cure of a glioma, it is not strange that at once doubts are raised as to the diagnosis. For it is well known that the clinical picture of glioma may occasionally be reproduced by products of inflammatory processes. Such objections have, however, no place in our case. I need not even refer to the anatomical findings in the first eye. For even the ophthalmoscopic picture in the left eye did not permit of another diagnosis. The disease had not yet reached the stage at which it was possible to mistake a vitreous body exudation with its sequelæ (pseudoglioma) for it. In spite of contrary statements by other authors, Hirschberg is correct when, in his monograph on glioma of the retina (1869), he says that the ophthalmoscopical picture (of beginning glioma) is so characteristic that a confounding with other affections could hardly be thought of. Hirschberg was, also, the first to draw attention to the enlargement and tortuosity of the bloodvessels growing from the papilla towards the tumor nodules. The white, sharply defined nodules protruding from the retina with a perfectly clear vitreous body, not even containing traces of finest dust-like opacities, as are always seen in such retinal processes which might here come under consideration, with the numerous, large newformed bloodvessels, prove the neoplastic character of the process; and it could furthermore not be misunderstood because on account of the frequency of the existence of glioma in both eyes we had especially searched for it. The clinical correctness of the observation of a benign course for five years cannot be shaken. We can even speak of a retrogression of the tumor. For, the last examination showed a flattening of the newformations, a disappearance of the nodular projections, so that they now appear like choroiditic patches of older date.

What still projects into the vitreous body is composed of white crumbs—lime-crumbs as we know them—as well as regressive changes in glioma from histologically examined cases. We surely are not foolish enough to speak of a cure of this glioma, because we cannot know whether living tumor cells are not left behind which suddenly may begin a rapid growth again.

Similar cases of such a retrogression of a glioma in an otherwise undamaged eye are hardly to be found in literature. For in the cases with temporary retrogression this was without exception accompanied by shrinking of the globe. (Wintersteiner, De Klein, Kieper, Lindenfeld.) In Wintersteiner's book on glioma there is one case[6] especially mentioned, "because the retrogression appeared very early and its phases could be followed with the ophthalmoscope, and because the retrogression took place without shrinking of the globe." But in this case the retrogression was of short duration, and two months later the tumor began again to grow rapidly, to perforate and cause death. The more recent literature only contains a communicatiion on a case by Deutschmann,[7] in which in the secondly affected eye a tumor the size of a pea had in seven months not only not grown but almost totally disappeared, leaving few traces. In the other eye, which had been enucleated, a tumor was found which in the parts lying in the retina showed the typical structure of a glioma. The patient had been treated with tuberculin, energetic inunctions with mercurial ointment, injections of cacodylate of sodium and iodide. That even at the latest stage in a hitherto perfectly unexplainable manner a standstill in the process may occur is shown by the case of Kleijn, in which at the enucleation the optic nerve even was found to be infiltrated with glioma cells and yet no relapse occurred.

Most recently, Axenfeld[8] reported a retrogression, perhaps even the cure of glioma nodules in the retina in one case after intense irradiation with Roentgen rays; and it may, therefore, perhaps be possible that the two irradiations in our case had this rare effect. I am sorry I have no definite data as to the strength of the rays applied and other details of this treatment. There is certainly no damage to the skin or hair visible. Yet, there are no definite proofs of such a curative effect of the Roentgen rays in our case. For aside from the fact that the development of the tumor in the right eye progressed unabated and soon after produced an increase of intraocular pressure, no changes were seen in the nodules in the left eye in the months following the irradiation. Even the standstill in the growth is of no importance, since the nodules had existed probably for a long time and their growth had probably from the beginning been a very slow one. Furthermore, for a year after the irradiation the nodules

6. Grolman. Page 146.
7. Ueber atypisches Netzhautglioma, Z. f. A., XXVII, 1912.
8. Klin. Mtsbl. f. Augenhlk., January, 1915.

seemed to be rather larger than smaller and even two years later no particular change was observed. Had the irradiation caused a necrosis of the tumor cells, the picture should have become altered earlier through atrophy of the tumor bloodvessels, although we must state that the ophthalmoscopic aspect of the nodules need not have been changed very greatly even by such a necrosis. The deposits of lime would probably, also, have been visible at an earlier date. I, therefore, believe that the favorable course in our case was not due to the irradiation with Roentgen rays, but to that absolutely unknown factor which in very rare cases produces a sudden dying out of the neoplastic process, even when the disease is far progressed. The history of malignant newformations of any kind contains such very wonderful examples of an unexpected cure.

A CONTRIBUTION TO THE STUDY OF BITEMPORAL HEMIANOPSIA WITH NEW INSTURMENTS AND METHODS FOR DETECTING SLIGHT CHANGES.

Clifford B. Walker (*Archiv. of Ophthalmology*, July, 1915) comes to the following conclusions:

(1) In general, field examinations are not complete unless disks representing a large range of visual angles (from 1.7 degrees to 8 degrees) are used according to the requirements of the case. (2) Field defects found to the normal disk should be examined with a larger series of disks to determine as much as possible concerning the prognosis and status of atrophy or optic block. (3) Cases showing no defect to the normal disks should be examined with the smallest series of disks as a test for the earliest changes for diagnostic purposes. (4) Temporal islands of vision occur in these cases on and below the horizontal line in the region of 30 degrees to 50 degrees from the center; these islands probably represent the strongest part of the temporal field and add encouragement to the prognosis. (5) Response to large disks in a defective field is also of encouraging prognosis. (6) Cæco-central regions are the weakest part of the field, and field defects usually extend vertically through this region, more marked, as a rule, from above than from below. (7) Scotomatous cases may be the result of the hypersensitive macular or cæco-central fibers exposed to toxic as well as pressure effects of the growth.

IS THERE A SYMPATHETIC AMBLYOPIA?*

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In Schirmer's[1] comprehensive work on the sympathetic eye affections, the nature of sympathetic irritation is explained in such a way that by the irritation of the centripetal nerves in the first affected eye an abnormal irritability, often combined with diminished functional capability, may occur in the area of all the centripetal and centrifugal nerves of the eye. There may appear, also, disturbances in the sensory apparatus, consisting of a diminished function or of increased irritability, which may grow to the appearance of spontaneous subjective light perceptions.

Schirmer then adds: Among the conditions of depression sympathetic amblyopia arouses our greatest interest, a diminution in central visual acuity combined with a concentric reduction of the visual field, in rarer cases with scotomata; and he warns against confusion with sympathetic asthenopia which in many cases is due simply to a weakness of the accommodation, but may also be of retinal origin.

In looking over the then reported cases of sympathetic amblyopia, Schirmer found such an unclearness in the outlining of the disease picture that after having tabulated 48 cases for a critical sifting he found only 25 which satisfied his demands. These demands are that this term must be reserved for a purely functional weakness, which is caused by irritating conditions in the other eye and stops with their disappearance.

For this reason Schirmer excludes Cohn's two cases (Schirmer's table 7 and 8) because, certainly in one case, probably in the other, there was simulation, while he accepts one case of Brecht[2] (table 13) although the restitution of sight did not follow at once, but exceptionally slowly after enucleation. A case of Yvert's appears dubious because the description is not complete; certainly this case cannot be cited as a proof. Furthermore, we agree with Schirmer without hesitation when he excludes the cases of Krenchel,[3] Roosa,[4] and Rosenmeyer,[5] because there was optic atrophy, which was present, also, no less than seven times in Nuel's[6] sixteen cases. The remaining nine cases Schirmer does not accept because either the first eye was free from any irritation or was even not present, or because the enucleation exerted no influence whatever, and some of the cases were but unsatisfactorily observed.

*Klin. Mtsbl. f. Augenhlk., Vol. LIV. page 250.

We can, therefore, by right exclude 22 of the 48 cases which Schirmer collected. If we further deduct Brecht's case, which Schirmer calls dubious, only 25 cases remain from which Schirmer formulates the following disease picture.

The first affected eye is painful and irritated, also, in a condition which proves an irritation of the ciliary nerves, although an inflammation is not always certain. Not at all regularly phthisis bulbi was present; neither had there been always an injury. The time between the affections of the two eyes was usually several years. Sometimes before the sympathetic amblyopia appeared a new condition of irritation had come on.

The diminution of the visual acuity in the other eye is usually only moderate and coincides at times with exacerbations of the irritation in the first eye; in other cases a concentrical contraction of the visual field occurs and scotomata are said to have been observed. Although the type of tiring, as described by Wilbrandt-Foerster, was not sought for, Schirmer does not doubt that it exists. The irritation depends on that of the first eye, and was mostly cured by enucleation, in Cooper's case by iron and quinine. The rapid, sudden return of the visual acuity is characteristic, so that for this reason alone Brecht's case, above mentioned, appears suspicious. Of the two cases of Maats and Vignaux[7] (table 3 and page 17), which Schirmer considers as especially proving his standpoint, I shall speak later on.

This opinion of Schirmer's has for years been the reigning one, and thus sympathetic amblyopia has taken its established place in ophthalmologoy. However, there were not wanting those who doubted the independent existence of this disease picture, even that it at all was a form of sympathetic irritation. These doubts Peters, too, shared for many years, since in his ophthalmic work of thirty years he has never seen a case of sympathetic amblyopia, but amblyopia with irritation of the first eye, which was explained in another way. It seemed, therefore, not improper to examine the 25 cases which Schirmer had considered as confirming, as well as the material published since Schirmer's publication, as to whether the assumption of a sympathetic amblyopia is really justified. Peters[8] gave expression to this opinion two years ago in a clinical lecture on the diagnosis and therapy of sympathetic inflammation in these words:

"Many cases of sympathetic amblyopia of the older literature are certainly nothing else than a true sympathetic irritation complicated by hysterical symptoms. In favor of this speaks above

all the contemporaneous appearance of concentrical contraction of the visual field—further symptoms of nervous asthenopia, which itself is nearly related to hysteria. It may easily be understood that a patient afraid to lose his second eye gets into a state of nervous irritation which lets him exaggerate all his troubles and fall under all sorts of suggestive influences. Thus these cases assume an especial importance in a forensic direction, since, aside from the sympathetic inflammation, the question of a traumatic general neurosis has to be considered."

Applying these criteria to the existing material, we find the following:

I. In Cooper's case (Schirmer's tables, case 1) it is stated that the first eye was phthisical and the second one irritated and had photophobia. The diminished visual acuity was improved with iron and quinine and the irritation disappeared, which is apparent since the second eye is spoken of as cured.

In this case, according to my opinion, every proof for the presence of sympathetic amblyopia is wanting; and I must say right here for a whole series of the later cases that a photophobic, irritated eye always shows a diminution in visual acuity when, in consequence of the irritation, the tear flow is somewhat increased. Every increase in the layer of fluid spread over the cornea causes a diminution of vision. If in Cooper's case a cure was obtained with iron and quinine, this corresponds to the manifold experiences of Peters, who for a long time has used a combination of secale cornutum, iron and quinine, for independent neuralgic conditions of the eye and its adnexa.

The case of Cooper can, therefore, not be called a confirming one.

II. The same holds good in the case of Pagenstecher (Schirmer's tables, case 2), in which the diminution in vision was contemporaneous with exacerbations in the irritation of the other eye. Aside from the assumption that the retinal veins in the second eye had been tortuous, there existed an asthenopia. Since after a week vision was again normal, this diminution can be brought into relation with the irritation, and we must not forget, as Schirmer, too, states, that in sympathetic amblyopia on account of the rapid tiring out of the eye, measuring the visual acuity is mostly a very difficult feat.

III. In case III. (Maats, Schirmer's table, case 3 and p. 17), which is brought forward by Schirmer as a paradigma of sympathetic amblyopia, the second eye, also, was injured a few weeks

after the injury to the first. It got red, became photophobic, painful, and teared continually. The patient who "believed" he could no longer see even larger objects, visited a physician only two years later who enucleated the shrunken, painful first eye, with the result that in two hours after the awakening from the narcosis all symptoms had disappeared and vision was normal.

In this case the patient evidently "believed" that he could not perceive larger objects, which certainly suggests a psychical sympathetic irritation the existence of which cannot, from the data given, be doubted. Pfalz considers this to be a case of traumatic neurosis.

IV. Mooren's case (Schirmer's tables, case 4) is one of leucoma adhærens after ulcus corneæ.

In the second eye the visual field was much contracted and objects were dim. There was lacrimation, photophobia, ciliary neuralgia and slight conjunctival injection. After this condition had existed for a year, the dim vision was suddenly cured; three weeks later the field was normal. In this case the concentric contraction of the field points to a functional disturbance which complicated the sympathetic irritation and might very well have existed independently of it. Whether the dim vision (Nebelsehen) and amblyopia can directly be identical, we doubt, since such dim vision is usually due to a disease in the eye.

V. and VI. In cases 5 and 6 (Schirmer's tables, 5 and 6), also reported by Mooren, the visual field was, also, considerably contracted concentrically. In the one case the accommodation was reduced and only No. 7 could be read. After an iridectomy the accommodation was at once improved and three weeks later vision and field were normal. Aside from the fact that we cannot understand how an iridectomy could at once improve the irritative condition in the first and second eye, the amblyopia is not definitely proven in the first case, since being able to read only larger type may be explained by the reduction of the accommodation.

Four further cases of Mooren's[9] cannot be admitted to be more confirming.

VII. In the first case (Mooren) the sympathetic neurosis of the second eye had been present for 28 years, and visual acuity was reduced to one-half. A few days after enucleation patient was perfectly cured. This case is too insufficiently reported to be used as a proof of sympathetic amblyopia.

VIII. In case 10 (Mooren) there was not even a diminution

in vision, only contraction of the visual field, photophobia, accommodative disturbances, which symptoms also disappeared after enucleation.

IX. The following case (Mooren) is described much more carefully. After a contusing trauma the patient suffered from purulent cyclitis, the other eye showed vision only one-fourth and concentric contraction of the visual field. In this case the restitution of vision and field took place within two weeks, which, as Schirmer says, too, does not exactly speak for sympathetic amblyopia.

X. In the fourth case (Mooren) vision was reduced, also, to one-fourth and the field narrowed. After enucleation vision rose to one-half. It is not rare that such changes in vision are seen in astigmatic eyes, on the other hand the co-existence of amblyopia and narrow field points to a functional neurosis.

In the case of Brecht,[2] already mentioned, one eye had become pththisical, but was no longer irritated. About five years later diminution of accommodation appeared, also contraction of the field and reduction of vision to 15/70. After enucleation, made 10 months later vision rose to 3/4 and the field became normal. Schirmer thinks this case doubtful on account of the slowness of the improvement. I believe that it should be rejected, because there is not a trace of a proof of a sympathetic affection, since the first eye was free from all irritation, and we may well question what would have been Brecht's diagnosis had the first eye been normal. Then, according to present knowledge, we would have to search for a functional neurosis and would have to explain why vision rose to 3/4 only and not to the norm.

Thus we come to Vignaux's[7] twelve cases, published in 1877.

XI. In case 13, a 14 years old girl had, 10 years after an inflammatory affection, spontaneous pains and pain on pressure in the phthisical eyeball and symptoms of sympathetic irritation in the other eye with such loss of vision that she had to be led about. After enucleation of the painful stump vision rapidly improved and a year later was still good. Since all exact data concerning the visual acuity and eventual material changes in the second eye are wanting, the loss of vision can probably be explained by the sympathetic irritative conditions, and we can say at most that with the disappearance of the sympathetic irritation the eye could be used better for seeing. The proof for the direct dependence of the loss of vision on the other eye is not clear.

XII. In case 12 (Vignaux's case 44), in which photophobia, lacrimation, and ciliary neuralgia were present in the second eye, vision rose from 3/20 to 14/20 within two weeks after the enucleation of a shrunken irritated eyeball, an improvement which is fully explained by the cessation of the lacrimation in an irritated eye. We must, further on, reckon with the possibility that other organic changes were present, causing the loss of vision.

XIII. In case 13 (Vignaux's case 46) the enucleation removed photophobia, lacrimation and ciliary neuralgia in the other eye. V.=5/15 before the operation, improved continually. These data, too, are too meagre to base a diagnosis of sympathetic amblyopia on them.

XIV. The same holds good in case 14 (Vignaux's case 56), in which after 20 days vision had risen from 1/8 to 1/3. Aside from the slowness of the improvement after the operation, we must ask what has became of the other 2/3 of vision after the cure.

XV. In case 15 (Vignaux's case 60) a general improvement of vision is reported. Patient could finally read again after the previous lacrimation was stopped.

XVI. In case 16 (Vignaux's case 67) no figures are given as to the loss of vision, which is said to have been considerable. There were, however, lacrimation, photophobia and ciliary neuralgia. Here, too, the lacrimation must be granted a certain importance in the loss of vision.

XVII. In case 17 (Vignaux's case 71) an improvement of vision is stated after it had sunk to 2/7. The data are insufficient.

XVIII. In case 18 (Vignaux's case 74) vision was so poor that patient had to be led. It is stated that all signs of sympathetic irritation were present and a rapid improvement followed the enucleation.

XIX. In case 19 (Vignaux's case 80) there were photophobia, lacrimation and pain on pressure. Vision rose from 1/3 to 5/7 without an explanation why it did not get normal.

XX. Case 20 (Vignaux's case 82) is remarkable because coincidently with relapses in the other eye the loss of vision was observed in an eye which was, also, suffering from photophobia, lacrimation and weakness of the accommodation. Since here a rapid cure with improvement of vision to the norm was obtained, we are forced to assume that the lacrimation interfered with good vision.

XXI. The same must be said as regards case 21 (Vignaux's case 83).

XXII. Case 22 (Vignaux's case 85) is again an example of insufficient foundation for the reduced vision after the cure, for V.=7/15 before and 14/20 after enucleation does not prove the exclusive influence of sympathetic irritation.

XXIII. The case of Landesberg,[10] which Schirmer accepts, I doubt, because in the second eye the papilla was pale and there was a central scotoma. These alterations have nothing to do with functional disturbances and must be looked upon as an organic affection of the optic nerve.

XXIV. In Chisolm's case (Schirmer's tables, case 30) it is especially stated that there was an eye shrunken after panophthalmitis, but free from irritation, and that some time after the enucleation vision had become normal; therefore, there was neither sympathetic irritation nor a sudden improvement after enucleation.

XXV. Finally we may mention Greef's[11] case, in which the contracted visual field became larger when the irritated eye was closed, the vision of the eye also rose from 6/9 to 6/6.

I should like to say to this case that such changes are, also, met with during the examination of vision when covering non-irritated eyes, and, I may add, that suggestion here may play a role, and, furthermore, that differences in reading test letters from 6/9 to 6/6 are of daily experience.

In reviewing shortly these cases, which Schirmer considered as proofs for the existence of a sympathetic amblyopia, we are struck at once by the fact that in a whole series of these cases a severe irritation existed in the so-called amblyopic eye, which among other things was characterized by lacrimation. Furthermore, this irritation combined with lacrimation has sometimes appeared with exacerbations in the irritation of the first eye, and thus these cases, too, will have to be judged in the same manner.

It is a daily experience that when we test the visual acuity of an eye which is irritated, and especially when it tears, the tests are unequal and vision is deteriorated. If these cases are not combined with contraction of the visual field, there is a mechanical and not a functional amblyopia caused by a momentary irritation of the optic central apparatus.

In another series of cases there is a concentric contraction of the visual field with or without deterioration of vision, and in not one of these cases has the attempt been made to exclude a

functional neurosis like hysteria. Of course, most of these cases come from a time in which the importance of concentric field contraction, and especially the subsequent field enlargement, as hysterical symptoms were not fully understood. Anyhow, these cases cannot be considered as confirming ones, since a coexisting functional neurosis cannot be excluded. And there is another point: of these 25 cases not less than 19 have been reported by 2 authors (Mooren 7, Vignaux 19). The question immediately arises whether these authors have not searched for these symptoms of diminution of the visual acuity and made use of everything they could gather together? May there not be some autosuggestion at play or inaccuracy in finding sources of mistakes? And, even, if we add to these two the other six authors, there were on the whole globe just 8 oculists who were graced by being able to see once or several times cases of sympathetic amblyopia, while from all the others this was withheld! From the very scarcity of such a coincidence of diminution of visual acuity in the sympathetically irritated eye we should conclude that it would be best to be very careful in assuming a causal relation in the sense of a marked functional amblyopia. We should rather think of a complication with general neurosis than of a functional disturbance of the optical central apparatus produced by the eye.

When we see how the teachings of sympathetic amblyopia have been further developed, we must at first draw attention to a paper not mentioned by Schirmer, by Kirst,[12] which is characterized by a certain lack of judgment.

The first case intended to prove the existence of sympathetic amblyopia is one of corneal trauma followed by traumatic cataract, which was absorbed in three years after the injury; two years after the injury the previously normal vision in the other eye was still normal, then it was reduced to 5/15. Besides slight corneal opacities which had not affected vision previously, there were a few floccules in the vitreous body. Since there was not a trace of irritation in the first eye, we cannot see why the amblyopia in the second eye should be due to sympathetic irritation. There was, also, a color scrotoma, *eo ipso* a functional disturbance.

In the second case reported by Kirst an eye was enucleated on account of secondary glaucoma, in 1895. A year later the orbit, infra- and supraorbital nerve were painful on pressure. The other eye had V.=5/20 and contraction of the visual field, also photophobia. The contraction of the field improved later, vision

rose to 5/6 and there was synchisis scintillans. The patient had chewed a great deal of tobacco. It is, further, stated that the papilla was somewhat pale. These conditions show that this case must be excluded.

The third case is as little convincing. In it there was, also, in the second eye a contraction of the visual field with normal vision, and the stump of the optic nerve after enucleation of the injured eye was free from irritation and not sensitive.

In the fourth case the stump of the optic nerve was sensitive. In the second eye the accommodation was reduced, the field slightly contracted and V.=5/10 with mixed astigmatism.

Kirst is perfectly conscious of putting himself into opposition to Schirmer in considerably extending the meaning of sympathetic amblyopia, but agrees that cases with optic nerve changes are to be excluded.

He differentiates between two categories of sympathetic amblyopia:

(1) One, in which vision returns to normal as soon as the irritating eye is removed. To these cases he counts the 25 of Schirmer.

(2) The other in which, in spite of the enucleation of the first affected eye, the amblyopia in the second is not improved, or in which the amblyopia appears only after the operation. To these cases he counts three of his own and the nine of Nuel.[6]

Kirst explains the sympathetic irritation to be a condition of irritation transferred by the trigeminus to the visual centre in which the optic nerve is not participating. Finally he acknowledges that hysteria might produce very similar symptoms.

After this it will be well not to consider Kirst's cases as proofs for the existence of a sympathetic amblyopia. We will, also, have to argue against Nuel's[13] description in 1904, in which in the picture of sympathetic amblyopia he lays stress on the fact that after contraction of the visual field a paleness of the papilla appears, as there were quite a number of affections of the optic nerves in the cases above mentioned. These cases must be excluded from our consideration.

The case of Consiglio,[14] in which there was paleness of the papilla and contraction of the field in the second eye, too, must be excluded, the more since in the only similar case of Rosenmeyer,[5] in which a retrobulbar neuritis was said to be the cause of the sympathetic amblyopia, the patient later on was proven to be a mallingerer.

There have but a few cases on this subject been published of late. Féjer[15] describes a reduction of the visual acuity to 1/70 and considerable concentric contraction of the visual field, in a 28 years old cook, with severe pains in the other shrunken eyeball, which disappeared after enucleation, vision returning to normal. Hence in this case, again, no attempt was made to exclude hysteria and since the second eye had, also, been afflicted with photophobia and lacrimation, we must have the same doubts about this case as about the others.

In the case of Weekers,[16] one eye was lost by a trauma. The other showed moderate photophobia and blepharospasmus besides contraction of the visual field and reduction of vision to 5/36. These are a series of hysterical symptoms, and a corresponding treatment led to a complete cure.

Weekers made a similar observation in three further cases in which, based on former communications, the diagnosis of sympathetic amblyopia might be made, while there was only a neurosis with predominating eye symptoms. In the discussion Nuel said that there are cases on the other hand in which symptoms of a neurosis are totally absent and yet an amblyopia exists.

A case of Elschnig's[17] is very instructive, in which the diagnosis of sympathetic amblyopia due to a trauma of the first eye seemed at first justified, but suggestive influences produced an improvement. Organic changes developed much later.

Considering sympathetic amblyopia, Elschnig is very sceptical and insists that with sensitive and irritable individuals the affection of one eye is alone able to disturb the vision of the other without the presence of a true neurosis, and he points out that there are, for instance, individual differences in the case of a corneal injury to which some do not react at all, while others react with a most severe spasm of the lids and photphobia in both eyes. Finally, simulation and aggravation must in all these cases be especially looked for.

If, furthermore, Speleers[18] wants to declare two cases of sympathetic amblyopia with central scotoma as belonging to sympathetic inflammation and not to sympathetic irritation, we can from what has been said not disagree with him.

The last case is by Perlmann,[19] who describes it extensively after having shown the patient at the meeting of the "Verein rheinisch-westfaelischer Augenærzte."

There was a wound in the cornea which reached beyond the corneal margin followed by traumatic cataract. The other eye

had normal vision. Sixteen and one-half months later vision was said to have become gradually considerably reduced. There was photophobia and lacrimation, congestion to the head, slight nervousness, fear, beating of the heart, loss of appetite and a general psychic depression.

Careful neurologic examination showed a slight degree of weakness of the whole nervous system. The injured eye was sensitive to light; the other teared, was photophobic, the visual field was slightly contracted, vision had sunken to 5/25, and the pupil, while reacting well to light, was immovable in accommodation and accommodation was paralyzed.

After the enucleation the conditions of irritation gradually subsided, yet the weakness increased, vision fell to 1/10 and the paralysis of the accommodation remained unchanged. Antiluetic and roborating treatment produced but a slight improvement. Vision rose to 1/5, the field grew larger, the color fields remained small as before, the accommodation remained unchanged.

Later neurological examination did not reveal hysteria. The patient improved generally, but 4 years after the injury vision was practically the same.

We agree with Perlmann, that up to the enucleation undoubtedly a sympathethic irritation was present, the symptoms of which (photophobia and lacrimation) disappeared after the enucleation, while loss of vision, weakness, contraction of the visual field and paralysis of the accommodation persisted.

The diagnosis of traumatic hysteria Perlmann refuses, since no symptoms of hysteria were present if the field contraction was neglected, which was not concentric as is seen in hysterics in whom, also, changes in the limits of the field are observed.

A suspicion of simulation, he also refutes because paralysis of the accommodation and weakness of the sphincter cannot be simulated. Furthermore, the patient went to the physician only 16½ months after the injury, although she had suffered from these symptoms for a long time.

Finally, Perlmann pleads for a malignant form of sympathetic amblyopia, aside from a benign one, without, however, stating how he imagines this to come about.

Sattler,[20] in reviewing this paper, states rightly that in Perlmann's case a traumatic neurosis might very well have been present, for the paralysis of the accommodation and the lack of the sphincter reaction on the attempt at fixation might well be explained by the lack of will impulse to accommodation, and

thus the reaction of the pupil to light would, also, be explained.

I am inclined to accept this explanation and, furthermore, to point out that if a paralysis of the accommodation cannot be explained by other causes this does not give the right by exclusion to assume its origin by way of sympathetic irritation, for there are cases enough of one-sided paralysis of the accommodation in which the ætiological factor cannot be found.

If, furthermore, Schirmer acknowledges sympathetic amblyopia only when after enucleation vision returns to the norm, Perlmann's case from this standpoint, too, is not one of sympathetic amblyopia.

I see in this case a paradigma, for the fact that the symptoms of sympathetic irritation are promptly removed by enucleation and if thereafter visual disturbances of varying degrees remain, the proof is given that in this special case they must be explained not by local but by a general affection.

Finally, a paper by Pfalz[21] on sympathetic irritation may here be mentioned, who is very sceptical as regards sympathetic irritation in general, and states that by irritation of sensitive nerves (not necessarily the trigeminus) momentary amblyopia and even amaurosis and contraction of the visual field may be produced. However, Pfalz does not go into details to prove his contention, and it seems doubtful whether such occurrences are possible, when no hysteria is present.

If we now base our further observations on the whole material found in the literature on sympathetic amblyopia, we see that the 25 cases formerly accepted as proofs of its existence by Schirmer, cannot withstand a serious criticism, and that in the more recent literature no case can be found which could alter this opinion. On the contrary, the cases of Weekers prove drastically that the suspicion uttered by Elschnig and Pfalz was well founded, that functional disturbances in the central nervous system play here an important role, let alone the possibility of simulation. And if we did not feel like refusing Perlmann's case at once, we should certainly ask whether one case can alone be sufficient to prove an independent disease entity.

It seems to me to be rather the right moment to strike the independent disease picture of sympathetic amblyopia from ophthalmic literature. The cases in which after enucleation vision promptly improved, can be explained mostly by the disappearance of the symptoms of irritation (photophobia and lacrimation), symptoms which interfere mechanically or functionally with

vision. Or there may be suggestive influences which induced a rapid improvement of the amblyopia and the simultaneous contraction of the visual field.

We must not forget that when we propose the removal of a painful eye to a patient in order to remove present suffering or to prevent serious consequences to the other eye, we usually assure him that thereafter everything will be well, there is a strongly suggestive effect in this which we must not underestimate.

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JOINT REPORT OF COMMITTEES

Of the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the Academy of Ophthalmology and Oto-Laryngology,

Regarding the Establishment of a Joint Board to Arrange, Control and Supervise Examinations to Test Preparation for Ophthalmic Practice.

During the past year the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-Laryngology have considered and adopted the reports of their respective committees recommending that graduate courses in ophthalmology representing at least two years of work subsequent to taking the degree of Doctor of Medicine be established in medical schools of the first class; and that such work be recognized by conferring an appropriate degree upon those who have successfully completed it.

It seems clear that there is unanimous agreement as to the need for systematized and standardized training of those who are to practice ophthalmology. At least five of the medical departments of important American Universities have now arranged for such courses leading up to special degrees. But already it is clear that the number of graduate students who will take the complete course leading to such a degree will, in the near future, be small. Even in universities capable of furnishing facilities adequate for the training of large numbers, the great majority will not meet the requirements for the higher degree.

It is desirable that the standard of attainment for which university degrees are given should be kept fully up to the present standard of our best universities. As matters now stand, therefore, a large majority of those entering upon the practice of ophthalmology will not be reached or directly influenced, by these standards. It is extremely desirable that all who take up ophthalmic practice as a specialty should be induced to pursue systematic courses and to show proficiency therein.

Professional opinion, particularly the opinion of those who have already established themselves in ophthalmic practice, can bring about the desired change to a large extent, without assist-

ance from legal enactments and without the necessity for establishing new educational institutions. It is only necessary to bring into existence a competent body to outline the proper course of study to be pursued; to examine and pass judgment upon existing institutions that offer opportunities for such study, and when individual students have proved that they have profited from such opportunities, and have prepared for ophthalmic practice, to give them the advantages of a certificate to that effect. To accomplish this purpose, in which we believe the ophthalmologists of America are fully united, we make the following recommendations, unanimously agreed to by the joint committees representing the three organizations above named:

1. That by the conjoint action of the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and the American Academy of Ophthalmology and Oto-Laryngology, a Board be established to arrange, control, and supervise examinations, to test the preparation of those who design to enter upon the special or exclusive practice of ophthalmology.

2. That this Board consist of nine members; three to be chosen by each of the above named organizations, in the same manner as their presiding officers are named. At the first election each organization shall choose three members, one for three years, one for two years, and one for one year; and thereafter one each year to serve for a term of three years. Vacancies shall be filled for the unexpired term by the Society from which the preceding member has been chosen. No member of the Board shall serve more than six years continuously.

3. Members of the Board shall serve without compensation, but shall be reimbursed for actual expenses while engaged in the work of the board, provided all other necessary expenses of the Board and its appointees have been properly provided for.

4. The Board shall appoint from its own membership, and from the medical profession outside its membership, a sufficient number of learned and skilled examiners who shall conduct the said examinations and report thereon to the Board.

5. The examinations may be held in any city of the United States where good facilities may be obtained for conducting clinical and practical examinations.

6. The Board shall fix requirements to be met by all candidates for examination, which shall include the successful completion of a course in medicine in a medical school of recognized good

standing, at least two years before the examination; adequate study of ophthalmology and allied subjects; and payment of an examination fee to be fixed by the Board. It shall be authorized to prepare lists of medical schools, hospitals and private instructors recognized as competent to give the required instruction in ophthalmology.

7. Each candidate whom the examiners report as having successfully passed the required examination shall receive by the authority of the Board a certificate or diploma, setting forth this fact, but conferring on the recipient no academic degree.

8. The American Ophthalmological Society and the American Academy of Ophthalmology and Oto-Laryngology shall from the year 1920 require every candidate for membership in those bodies to possess the certificate above mentioned, unless the applicant shall possess a degree in ophthalmology conferred by a university recognized by them as competent to prepare its students for such a degree. The Section on Ophthalmology of the American Medical Association, in so far as it is empowered to adopt its own rules, shall from the year 1920 require that its officers and those members accorded places on its program shall possess the certificate in question or its equivalent; and shall request that in the directory published by the Association, the holders of such certificate be especially recognized.

GEORGE E. DE SCHWEINITZ, Philadelphia.
MILES STANDISH, Boston.
SAMUEL D. RISLEY, Philadelphia,
JOHN E. WEEKS, New York.
WILLIAM H. WILDER, Chicago.
HIRAM WOODS, Baltimore.
WALTER R. PARKER, Detroit.
ALEXANDER DUANE, New York.
WILLIAM ZENTMAYER, Philadelphia.
WENDELL REBER, Philadelphia.
WALTER B. LANCASTER, Boston.
EDWARD JACKSON, Denver.

ABSTRACTS FROM MEDICAL LITERATURE.

By W. F. Hardy, M.D.,
St. Louis, Mo.

THE SIGNIFICANCE OF THE PUPILLARY REACTION TO EPINEPHRIN.

The following is an editorial taken from the *Jour. of the Am. Med. Ass'n*, June 5, 1915:

"When a solution of epinephrin is instilled into the conjunctival sac, no noteworthy effect is produced on the pupil of the eye. After complete excision of a superior cervical ganglion of the sympathetic nervous system, however, similar introduction of epinephrin causes a dilatation of the pupil on the operated side. This was demonstrated by Meltzer at the Rockefeller Institute for Medical Research in 1904. The degree of pupillary dilatation depends on the quantity instilled. It can easily be made maximal and may last for some time. The miotic effect of physostigmin is also overcome by instillation of epinephrin. The dilatation of the pupil is accomplished by the contraction of a muscle, the dilator pupillæ, just as the constriction of the pupil is accomplished by the contraction of the constrictor pupillæ. These muscles are exact antagonists. When one is contracted the other is relaxed; and it seems to be an established conclusion that the relaxation is an active one, so that the changes noted at any time can take place promptly, rather than gradually. According to Meltzer's view, the normal effect of the superior cervical ganglion is to inhibit the dilator and to stimulate the constrictor and the effect of suprarenal extract is just the reverse of it, that is, to excite the dilator and inhibit the constrictor. Epinephrin can, therefore, show its proper effect only after the antagonistic activity of the ganglion is removed.

"We have reviewed these physiological investigations in some detail in order to afford an interpretation of the observation made by Loewi, in 1908, that instillations of epinephrin produce dilatation of the pupils in animals after the excision of the pancreas also. Loewi based an explanation of this mydriasis on the facts already cited. He assumed that the pancreas exerts an inhibitory influence on the sympathetic system so that the removal of the gland would bring about a situation comparable to that noted after extirpation of the cervical ganglion. After either

procedure the introduction of epinephrin into the conjunctival sac can induce mydriasis. Loewi attempted a practical application of these phenomena by examining the pupillary responses of persons suffering from various diseases, particularly diabetes. The discovery of positive mydriatic reactions after epinphrin instillations in several of these cases suggested this as an indicator of pancreatic involvement. The reaction has accordingly been proposed as a test for pancreatic disease and particularly diabetes of pancreatic origin. It is now reported by Loewi and Rosenberg that the mydriatic reaction of Loewi can be brought about in any condition in which there is an excess of sugar circulating in the blood. Not only can the characteristic pupillary response to epinephrin be produced whenever hyperglycemia results in animals after intravenous infusions or even liberal ingestion of glucose, but it also ensues when the sugar level of the blood is raised by certain drugs. The Loewi phenomenon, therefore, cannot be correlated solely with the pancreas, except on the improbable assumption that this gland becomes involved in every condition of hyperglycemia. The reaction can no longer be regarded as a characteristic test of pancreas function.

"In the hypoglycemia produced by phlorizin, the mydriatic response to epinephrin cannot be obtained, despite the prominence of the resulting glycosuria. Perhaps we may now assume that an excess of sugar in the blood directly affects the sympathetic inhibitory factors. It remains to be determined by clinical studies whether the pupillary reaction can be used to distinguish between true diabetes with an attendant hyperglycemia and the glycosurias of renal origin in which the level of the blood sugar is not raised. The introduction of satisfactory clinical methods of blood analysis has to some degree rendered other procedures superfluous."

OPTIC NEURITIS CONCURRENT WITH WHOOPING COUGH.

E. Sherman Clouting (*N. Y. Med. Jour.*, May 22, 1915) reports a case of optic neuritis concurrent with whooping cough. He is careful to make it clear that he does not absolutely attribute it to pertussus. He mentions the possible ætiological factors in optic neuritis, viz., poisons in the blood such as produce a polyneuritis—alcohol, arsenic, mercury and tobacco. It occurs also as a sequel of any infectious disease, also in

chronic anæmia, pernicious anæmia, leucocythæmia and diabetes. By some rheumatism, gout and taking cold are reckoned as causative factors. Its common cause is pressure within the skull exerted by tumor, abscess or syphilitic exudations along the base or by pressure of fluids in the ventricles. The case reported by Clouting was that of his own son. There was a history of pertussus of unusual severity also a history of trauma, the child having struck his head against a hydrant while sledding. No cerebral symptoms indicative of tumor or meningitis were present. No motor palsies, no sensory symptoms save pain, no Babinski, Kernig's sign negative, no opisthotonos, the pupils were equal and reacted to light and accommodation. The diagnosis of a bilateral neuroretinitis was made by Dr. Sweet and confirmed by Dr. Hansell. Renal involvement was suspected and excluded. Recovery was rapid after the 18th day of acute illness. Vision at present, O.D.=20/100, O.S.=20/20, and is still improving. Clouting asks himself the question if the condition was caused by trauma or hæmorrhage or an inflammatory condition affecting the sphenoidal sinus. He also asks if the child had a meningitis. If so, all the cardinal symptoms were lacking. In discussing the case, Dr. Spilber suggested an encephalitis. He regards pertussus as capable of producing encephalitis.

A CASE OF BILATERAL OPTIC NEURITIS DUE TO SPHENOIDAL SINUSITIS.

A. A. Bradburne (*British Med. Jour.*, Jan. 16, 1915) adds another to the long list of case reports of this condition. The striking features according to Bradburne were the method of onset, the implication of the abducens nerve and the fortunate termination. As far as his researches have extended the last two features seem not to have been recorded before. (Sluder and Ewing have however, in the pages of this Journal, December, 1914, recorded case reports of abducens paralysis the result of ethmo-sphenoidal suppuration.) Bradburne's case report is given in full. The presence of sphenoiditis was inferred from the symptoms and history. Operative treatment was not carried out, and the sphenoidal empyema evacuated spontaneously accompanied and followed by a marked subsidence in the neuritis and a disappearance of the diplopia. The author states that his case presents several features of interest and some problems;

the weakness of the abductor, the lapse of time before the neuritis was definitely evident, the absence of pain and tenderness over the eyeball, and the primary implication of the sixth nerve. The implication of the sixth nerve assisted the localization of the seat of the trouble, which could hardly be elsewhere than at its situation on the body of the sphenoid where it traverses the cavernous sinus alongside the carotid artery. By direct extension forward the implication of the optic nerve can be explained, as the optic canal can be formed entirely by the bone forming the sphenoidal sinus, as shown by Onodi. Bradburne suggests that in his case that the sixth nerve may have had an abnormal relationship to the carotid artery in the cavernous sinus, and instead of lying external to the artery might have lain on its mesial aspect in contact with the bone. "This theory would explain why the occurrence of abducens paresis has not been recorded before." (As seen above, it has been recorded.) The localizing feature of this paresis excludes the posterior ethmoid cells, affections of which are a frequent cause of optic neuritis. The accompanying occipital pain is, in the opinion of Bradburne, attributable to implication of the sympathetic plexus around the carotid artery. He is led to this view by the character of the ptosis in his case, which was not due to a levator paresis but to a weakness of Müller's muscle, which is innervated by the sympathetic. "That the sympathetic was affected is confirmed by the aggravation of the ptosis under mental excitement despite the fact that the other classical and text book symptoms were not at the same time present." The trouble began with the sudden cessation of a rhinitis, and spontaneously recovered with a recurrence of the original cold in the head.

THE MEDICAL TREATMENT OF PITUITARY DISEASE.

Wendell Reber (*N. Y. Med. Jour.,* February 27, 1915) writes briefly on this subject. For fear that his article would suffer by abstracting, it is given below in full.

"Thyroid medication has now reached a fairly definite basis of understanding, but this, we believe, cannot yet be said for pituitary medication. While our ideas as to the composition of the anterior and posterior lobes, and the pars intermedia and their various functions, are clearer to-day than they have ever been, the effects produced by preparations of the various por-

tions of the gland have shown such wide variations that we are not in a position to lay down accurate indications as to method and dose. Moreover, the interrelations between the thyroid and the pituitary body are by no means settled. Meanwhile, the surgeon, guided by fairly accurate findings (such as the general status of the patient, the skiagraph, and study of the visual fields), no longer hesitates to attack the pituitary body by the intranasal or extranasal route. In some cases the brilliancy of result has more than surpassed expectations, while in others fatalities have supervened. Indeed, the mortality has become an important factor. In his last twenty-six cases, Hirsch, operating by the intranasal route (which is considered by many the safest method), reports three deaths or about one in nine (eleven per cent). This of itself should give us pause, the more so as a small number of cases have been reported in which organotherapy has brought about almost complete disappearance of the signs and symptoms of pituitary disorders. Waldeck, for instance (*Jour. Michigan State Med. Assn.*, 12, p. 417, 1912), reports a case of hyperpituitarism passing later into the picture of hypopituitarism. Operation was refused, but there was marked improvement in general health under prolonged administration of pituitary extract. The skiagraph showed enlargement of the sella turcica and absence of the right posterior clinical process. Recently De Schweinitz recorded a case in which brilliant results followed medication with pituitary preparations, marked improvement showing within a fortnight. Another case, studied for three years (by Clothier and Devitt, of Philadelphia), I have the privilege of mentioning here in only a preliminary way. The man, about thirty-five years of age, presented classic bitemporal hemianopsia, with phenomena of hyperpituitarism. The skiagraph showed enlargement of the pituitary body. Medication with thyroid was instituted without any noteworthy improvement. Pituitary whole gland was then added to the treatment, and in four months normal vision was restored with reestablishment to full limits of the form and color fields. The patient then went without any medication for two years, when he was found to have an incomplete vertical hemianopsia in the right eye and marked contraction of the form and color fields in the left eye. Treatment was again instituted this time with pituitary (whole gland) substance alone, but the expected improvement did not occur. Thyroid substance was then added, and within twelve months the usual fields again re-

turned to normal limits for field and color. This last recovery dates back ten months, and the patient still enjoys full vision and visual fields. Pituitary disease is a relatively common malady which has been heretofore largely overlooked. There is no question that from now on it will be much more frequently recognized. The question will then present itself, whether the treatment shall be medical or surgical. With a mortality of eleven per cent attending the surgery of the condition, it seems that organotherapy should first be resorted to. And even though it may have to be pluriglandular, the end sought will more than justify the means. Eventually we shall learn whether thyroid or pituitary preparation or both are indicated, and when and how. Meanwhile we plead for a trial of organotherapeutics in pituitary disease. Should the medical treatment prove unavailing, resort may be had to surgery."

DISTORTION OF THE VISUAL FIELDS IN CASES OF BRAIN TUMOR.

Harvey Cushing and Clifford B. Walker contribute a monograph on this subject, which has been published in *Brain*, Vol. 37, March, 1915. The text takes up 60 pages and the following is a recapitulation:

In a series of 454 cases classified as tumor of the brain there have been 101 in which the lesion was hypophysial or parahypophysial origin, and in 81 of these cases chiasmal involvement lead to deformation of the fields of vision. These deformities tended at the time of admission to be bitemporal in 26 cases, homonymous in 12, were unclassifiable in 8 cases, and the remaining 35 showed blindness in one or both eyes, making it difficult to tell in which group—bitemporal or homonymous—they belonged.

In cases of bitemporal hemianopsia it is unusual for the field defects to be bilaterally symmetrical, the text-book representation of the condition, with a vertical meridian dividing blind from seeing retina in each eye, being relatively uncommon.

In the majority of cases, whether of intra- or suprasellar tumor, the first perimetric indication of the process is shown by a slant in the boundary of an upper temporal form field and a corresponding quadrantal defect in the color peripheries. For clinical convenience this is designated as Stage I. of the process, and with a progressive lesion the field defects advance in

characteristic fashion through Stage IV. of actual hemianopsia to Stage VIII., when blindness has ensued. In all stages the loss of color perception is usually in advance of that for form, and the condition in one eye is usually in advance of that in the other, so that every possible stage combination in the two eyes may be met with.

When relief from pressure has been afforded by operative measures the recession of the defect takes place in a sequence the reverse of that characterizing the stages of an advancing process. In the receding process relative paracentral scotoma often persist, as the functional vulnerability of the macular and paramacular fibers appear to be greater than that of the other fasciculi. Restoration of normal field peripheries is possible even when the process has advanced to or beyond Stage IV. of typical half vision, and some vision may be regained even after blindness has occurred, provided it has not been of too long duration.

In view of the prompt restorations in field outlines which may follow operative relief from pressure, it is evident that the so-called primary optic atrophy often does not represent an actual anatomical degeneration so much as a physiological block to the transmission of visual impulses.

In conclusion: Detailed perimetry with small test objects of serial sizes, particular attention being paid to the shading off of the upper temporal peripheries and to the presence of relative paracentral scotomata in the same quadrant, is advocated for patients with pituitary disease in order that stages of hemianopsia antecedent to those usually recognized may be detected.

BLASTOMYCOSIS OF THE EYELIDS WITH REPORT OF CASES.

Edward Jackson (*Jour. Am. Med. Assn.*, July 3, 1915) places on record two cases of a disease, as he states, hitherto supposed to be rare and given scant notice in ophthalmic literature and text-books. He calls attention to its clinical importance, the disastrous results, through distortion of the lids, of failure to recognize it and its excellent prognosis under proper treatment. The history of the affection is reviewed as is also the ætiology. The invading organism is supposed at times to find entrance through an injury. In both of Jackson's cases a history of trauma was lacking. This disease has been called the "Chicago disease," but

it has been reported from widely distant parts of the country. The disease seems to have a predilection for the lids. Wood concluded that one fourth of the cases affected the lids, and Jackson thinks this is not too high a proportion for the cases that are not systemic. In making the diagnosis, epithelioma, tuberculosis and syphilis are to be excluded. The diagnosis may depend on the miscroscopic findings of the organism. The clinical history and appearances of the case are sufficient to indicate its character with great certainty. The active lesion suggests epithelioma.

A striking feature of this condition is the great amount of resultant cicatrization. Except through involvement of the lids blastomycosis seems to have little tendency to affect the eyes. The chief therapeutic agent is potassium iodide given in doses of 30 or more grains three times a day, rapidly increased to the limit of tolerance. Operative measures are distinctly contra-indicated. The curetting of the lesions and the application of silver nitrate may bring about a condition favorable to healing, but the iodide is the essential agent. Copper salts and Roentgen rays have also been used with doubtful results. Attempts at vaccine treatment have been made with no definite results. The iodide is given on a purely empirical basis. The experience in systemic blastomycosis has tended to throw doubt on the specificity of the iodides, for the reason that many of these cases have gone on to a fatal termination in spite of very large doses of potassium iodide.

BLINDNESS INCIDENTAL TO EXTERNAL ETHMOIDAL OPERATION.

Harmon Smith (*Laryngoscope,* April, 1915) reports a case under the above title. The patient was operated upon for some nasal obstruction. An external ethmoid operation was performed on the left side under a general anæsthetic. He took the anæsthetic badly, bled profusely, so that but one side was completed. On removing the dressings the right eye protruded and the lids were greatly chemotic. Soon after this sight failed. No pain at any time. The work had been done by a rhinologist other than Smith. When sight failed an oculist, Dr. Reese, was consulted, who found marked chemosis, ophthalmoplegia externa, dilated pupil irresponsive to light. Media clear and nerve head showing neuritic atrophy with œdema in the surrounding retina. Upward and inward there was a large subhyaloid hæmorrhage.

The veins were tortuous and there was no perception of light. Dr. Smith found a polypoid inferior turbinate, deflected septum, ethmoiditis and sphenoiditis, and occlusion of both sides of the nose by a growth suspected of being sarcomatous but on examination proving to be myxomatous. Smith corrected the nasal condition and in doing so found a large perforation of the septum high up which had been made by the previous operator. This perforation went through the perpendicular plate of the ethmoid and the operator's instrument had evidently penetrated through this into the ethmoidal tract of the right side. It was assumed that the operator had lost his sense of direction. Smith could not decide whether the blindness was due to direct injury of the optic nerve or whether traumatism within the region of the nerve produced a hæmorrhage of the cavernous sinus, or whether pressure followed the induration of the tissues surrounding the nerve. The following points are emphasized by this case: (1) Removal of polypi incident to sinusitis should be done under local anæsthesia. (2) Orientation is maintained far better when the operation is performed under local anæsthesia with the conscious assistance of the patient. (3) With a patient under general anæsthesia, with the head in any other than a direct line, and with the operator working backwards and upwards, there is considerable danger of losing the direction.

BOOK REVIEW.

PRACTICAL MEDICINE SERIES. Volume VIII. The Eye, Ear, Nose and Throat. Edited by Casey A. Wood, Albert H. Andrews and W. L. Ballenger. 1915. Chicago: The Year Book Publishers, 327 S. La Salle St. Price, $1.50.

One of the welcome visitors especially to the general practitioner's office is this little volume which comes every year. It is needless to say that a year book compiled by men so well known in the profession is up to the mark in every respect. Like its predecessors, it should be in the hands of most, if not all, practitioners. ALT.